(Aus der internationalen Monatsschrift f. Anat. u. Phys. 1892. Bd. IX. Heft 7.)

On the Electromotive Phenomena of the Mammalian Heart [1])

by

W. M. Bayliss, B. A., (Oxon) B. Sc. (Lond.), and
Ernest H. Starling, M. D., (Lond.) M. R. C. P., Joint Lecturer on Physiology at Guy's Hospital.

(From the Physiological Laboratory of University College, London.)
Expenses defrayed by a grant from the British Medical Association.

(With pl. XV—XVII and 8 figs.)

Although the electromotive phenomena of the heart of cold-blooded animals has been the object of much attention and investigation on the part of physiologists [2]), those of the mammalian heart have, till recently, remained uninvestigated.

Waller and Weymouth Reid [3]) confined themselves to observations on the hearts of recently killed animals, and arrived at the conclusion that though, under certain conditions, the mammalian heart may show a diphasic variation indicating a wave of negativity starting at the base or apex, yet under normal circumstances it was probable that all parts of the ventricular muscle contracted simultaneously. This conclusion they drew from the fact that in hearts examined *immediately* after death the variation was monophasic. The authors believe that their results justify the conclusion that the mammalian ventricle is not only controlled by nerves, but co-ordinated as to the action of its several parts, through intravascular nervous channels.

---

[1]) This paper was communicated to the Royal Society on Oct. 23d 1891, and an abstract of it is published in the Proceedings. Vol. 50.

[2]) Especially Sanderson and Page (Journal of Physiology, II and IV.) For earlier literature, see their papers.

[3]) Phil. Trans. 1887.

C.

In a later paper by Dr. Waller alone[1]), he gives up the idea that all parts of the ventricle act simultaneously, and shews that the monophasic variations he obtained previously, were, in all probability, due to local injury. In the majority of cases in which he examined the electrical variation of the heart beating in situ (11 cases out of 17), he found the variation to be diphasic and shewing a wave of negativity starting at the apex and proceeding thence to the base. The greater part of the paper is taken up with the electrical variation of the heart in the intact animal to which, including that of man, we propose to return in an appendix to the present paper. Dr. Waller concludes that under normal circumstances the contraction begins at the apex and travels thence to the base, lasting longer at the base than at the apex. He leaves it an open question what the cause may be of the difference of the course of the wave in the hearts of frogs and mammals, though he mentions a suggestion that has been made to him that the contraction of the entire heart, commencing at the venous orifices of the auricles, is propagated thence by the auriculo-ventricular curtains and the musculi papillares to the apical vortex and thence upwards to the base of the ventricles. The paper also contains interesting observations on the effects produced on the form of the curve by injury at one or other of the leading-off electrodes, and which are similar to those found by Sanderson and Page in the frog's heart.

Frédéricq[2]) has also investigated the question by means of the capillary electrometer. He arrives at the conclusion that each cardiac contraction is a tetanus made up of fused contractions succeeding one another at the rate of 20 per sec. Most of his curves are monophasic (apex negativity), but in many there is a small initial, or terminal, phase denoting base negativity. He does not discuss the question as to whether there is a wave of contraction in the ventricle. A criticism of these results will be found below.

---

[1]) „On the electromotive changes connected with the beat of the mammalian heart, and of the human heart in particular." Phil. Trans. Vol. 180. 1889.

[2]) „Sur les phénomènes électriques de la systole ventriculaire." Travaux du Laboratoire. Liège. Tome II. 1887—1888. p. 133.

The intention with which we commenced the present work was three-fold:

I. To discuss whether there is evidence of a wave of contraction in the ventricle, and to determine its course and time-relations if present.

II. To obtain evidence as to the nature of the transmission of contraction from auricle to ventricle, chiefly by measurements of the time taken by the excitatory process in travelling from auricle to ventricle and from one point of the ventricle to another.

III. To examine Frédéricq's view on the tetanic nature of the ventricular contraction.

Method.

Our experiments were commenced on cats, but we soon found them unsatisfactory, the heart appearing to be more vulnerable than that of dogs, to which we afterwards confined our attention. In all cases the animals were anaesthetized with a large dose of morphia (2—4 grains) and chloroform. Tracheotomy was performed and artificial respiration carried on from the moment the chest was opened; sometimes curare was given, but as a rule it was not found necessary. A median incision was then made in the front of the chest, the sternum divided in the middle line by bone-forceps, and the two halves of the thoracic wall pulled asunder by hooks so as to give complete access to the heart. In opening the chest in this way, if one keeps to the middle line accurately, there is practically no bleeding. The pericardium was then opened by scissors and stitched to the thoracic wall on each side; in this way the heart was supported below and no longer affected by the movements of the lungs.

In some cases the vagi were divided before opening the chest, and prepared for excitation. As a rule the slowing of the heart produced by the morphia exciting the medullary inhibitory centre was found advantageous, — hence the vagi were usually left intact. Occasionally one of the carotids was connected with a manometer to register the heart beat, but in most cases the beats were registered by a small tambour pressing on the ventricle through the pericardium at the side. The heart being now exposed, two points of its surface were connected by nonpolarizable electrodes to the terminals of the

capillary electrometer. An image of the mercury meniscus was projected by means of a Zeiss C. objective and the limelight on to a slit, an image of this in turn being thrown on to a moving photographic plate by means of a short focus cylindrical lens. Between the lens and the plate were the lever of the recording tambour, and two chronographs, one giving a time-tracing of 8 or 100 per second according to the rate of movement of the plate, and the other being in the primary circuit of the induction coil when artificial excitation was used. Considerable difficulty was met with in finding suitable electrodes, since none of the ordinary forms will remain in contact with the heart during its vigorous alterations in shape. Brushes and threads altered too much in resistance as more or less of their length came in contact with the surface of the heart, and the twisting of the heart gradually displaced them altogether. At length we devised a modification of Hermann's nerve electrode, which answered the purpose very well (Fig. 1).

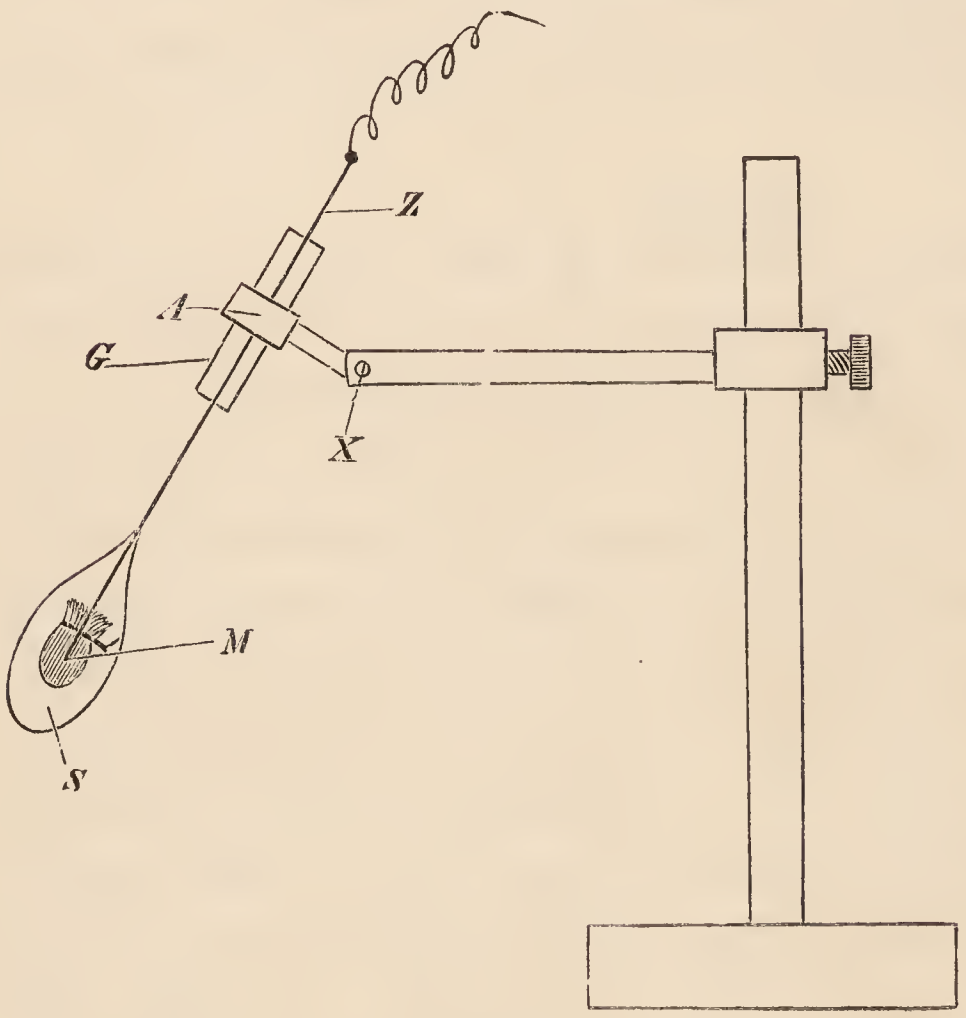

Fig. 1.

A zinc wire (Z) is well amalgamated at its lower end, which is surrounded by a small bag of muslin (M) containing zinc sulphate paste. Just before use a mass of normal saline paste (S) is put on around the bag of zinc sulphate clay, and moulded by the fingers to the form required. The zinc wire can glide freely in a glass tube (G) which is held in a clamp (A), itself freely moveable on an axis (X). The whole can be adjusted in height by sliding on the upright of the stand. These electrodes are so light that they follow the movements

of the heart perfectly, the adhesion of the clay to the surface of the heart being sufficiently great to overcome any tendency to jerking off produced by the momentum of the electrodes. They have a low resistance and are nonpolarizable and equipotential.

I. Excitatory wave in ventricle.

To determine whether there is a wave of negativity in the ventricle we led off from the various points of the outer surface of the ventricles. The electrodes were in most cases placed on the front of the right ventricle near the base, and on the apex of the left ventricle. We also led off from base and apex of left or right ventricle alone, and from points situated between apex and base. We find that in animals whose hearts are in as normal a condition as possible, *the variation is always diphasic, and shews negativity of the base preceding that of the apex.* The result is the same, whether the pericardium be intact or opened, or from whatever points of the heart's surface we lead off. (Figs. 1, 2, and 3. Plate XV).

This result is opposed to the majority of the results obtained by Waller as mentioned above. In our earlier experiments we obtained similar results to his, i. e. apex negativity preceding base negativity, though this was by no means an invariable result, our curves being often triphasic indicating base negativity followed by apex negativity and this again by base negativity. During this period of our investigation we were using air at the temperature of the room for artificial respiration; we thought, however, that the animal would remain in a more normal condition and the circulation be better kept up if we used warmed air, and for this purpose the air blown from the bellows was made to pass through a spiral tube surrounded by boiling water before entering the trachea. From the time that this proceeding was adopted we have always been able to rely upon getting a diphasic variation from the ventricle and always showing basal negativity preceding apical negativity. The conclusion we draw is, therefore, that in the mammalian heart as in the frog's heart the electrical change travels in the form of a wave from base to apex.

It was some time before we discovered the cause of the sudden

alteration in our results, from a variable apex-base or base-apex-base curve to a constant diphasic variation of the nature of a base-apex curve. Remembering then the effect of local cooling on the duration of the negative phase at the cooled point, described by Sanderson and Page, we thought it possible that the change might be due to the employment of warmed air, and in fact, this turned out to be the case.

The following experiment illustrates the sensitiveness of the electrical variation to changes in the temperature of the respired air.

June 17th. Dog. Operation as already described. Artificial respiration with *warmed* air.

Base of right ventricle to acid.

Apex of left ventricle to mercury in capillary.

Character of curves: —

a) Before opening pericardium.

Diphasic. Base — Apex.

b) After opening pericardium.

Diphasic. Base — Apex. (Fig. 4. Plate XV.)

The hot water was now poured out of the vessel surrounding the spiral tube and replaced by ice; after 5 minutes another photograph was taken and found to be triphasic.

c) Triphasic. Base — Apex — Base. (Fig. 5.)

After another five minutes:

d) Diphasic. Apex — Base. (Fig. 6.)

The ice was now removed and hot water replaced. After 10 minutes the variation was once more diphasic, the base becoming negative first.

e) Diphasic. Base — Apex. (Fig. 7.)

Cold air was then used again, with the same result as before.

We found that it was possible to imitate these results in a simple way by placing a lump of ice in close proximity to the base or apex of the ventricle respectively. Cooling the *base* by this means was found to have the same effect as the use of cold air in respiration.

When cooled air had been made to produce a variation indicating apex negativity preceding base negativity, it was found that the normal variation (base negativity preceding apex negativity) could be reproduced by cooling the apex with a lump of ice.

It is evident then that in using the cooled air in respiration, we are exerting by some means, a cooling influence on the base of the heart.

In the light of these experiments we can account for Waller's results on excised hearts. These were mostly allowed to get cold after extraction from the body, or when examined in situ they were no longer kept warm by the blood current. Now it is evident that the thin wall at the base of the ventricles would cool more quickly than the dense mass of muscle forming the apex, and the final result would be the same as using cooled air in the living animal.

In all cases of which mention has been made above, the ventricle is supposed to have been beating in normal sequence to the auricle; where this is no longer the case the contraction may probably start at any point in the ventricles, as Waller has pointed out, and generally from any spot that is warmer than the rest of the surface.

We have found that it is quite possible to obtain a normal diphasic variation from an excised h art. The heart must be beating well, and to ensure this we found it advisable to inject sufficient morphia to produce complete anaesthesia, and allow the animal to recover from chloroform, which is very apt to cause complete stoppage of the auricular beat; a moist chamber warmed to about 38° C. is prepared, the heart cut out as rapidly as possible and placed on a glass plate in the warmed chamber, electrodes applied to two points of the ventricular surface, the tambour arranged to shew the beats, and photographs of the variation taken at once. The whole process from the moment the chest is opened can be got through in not more than 30 seconds. We then get a diphasic variation shewing negativity starting at the base and followed by negativity of the apex. In some cases we found that while the right ventricle gave a „stepped curve" (see below) indicating partial cooling at the base, we could obtain the normal curve from the left ventricle. This illustrates well the effect of thickness of the ventricular wall on the rapidity of cooling.

We have now to consider the effects of warming or cooling different parts of the heart on the time-course of the electrical change. Sanderson and Page shewed that the effect of cooling any portion of the contracting ventricle of the frog was to increase the duration of the

negative state at that point, the rise and fall of the curve being rendered more gradual, and conversely the effect of warming was to shorten the duration of negativity and render more abrupt the rise and fall of the curve. Thus, if we consider the electrical changes at the exact points A and B only, we get the following result (Fig. 2). A and B are supposed to be at the same temperature.

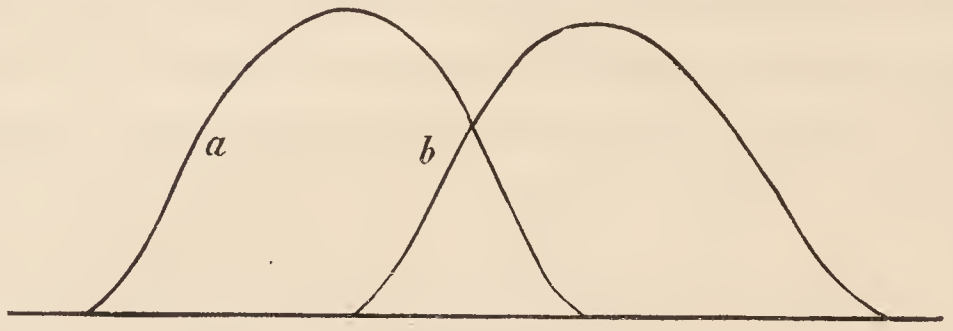

Fig. 2.

The curve marked a is the course of negativity at A, and that marked b, that at B. The effect of this on the electrometer will be the following. (Fig. 3.)

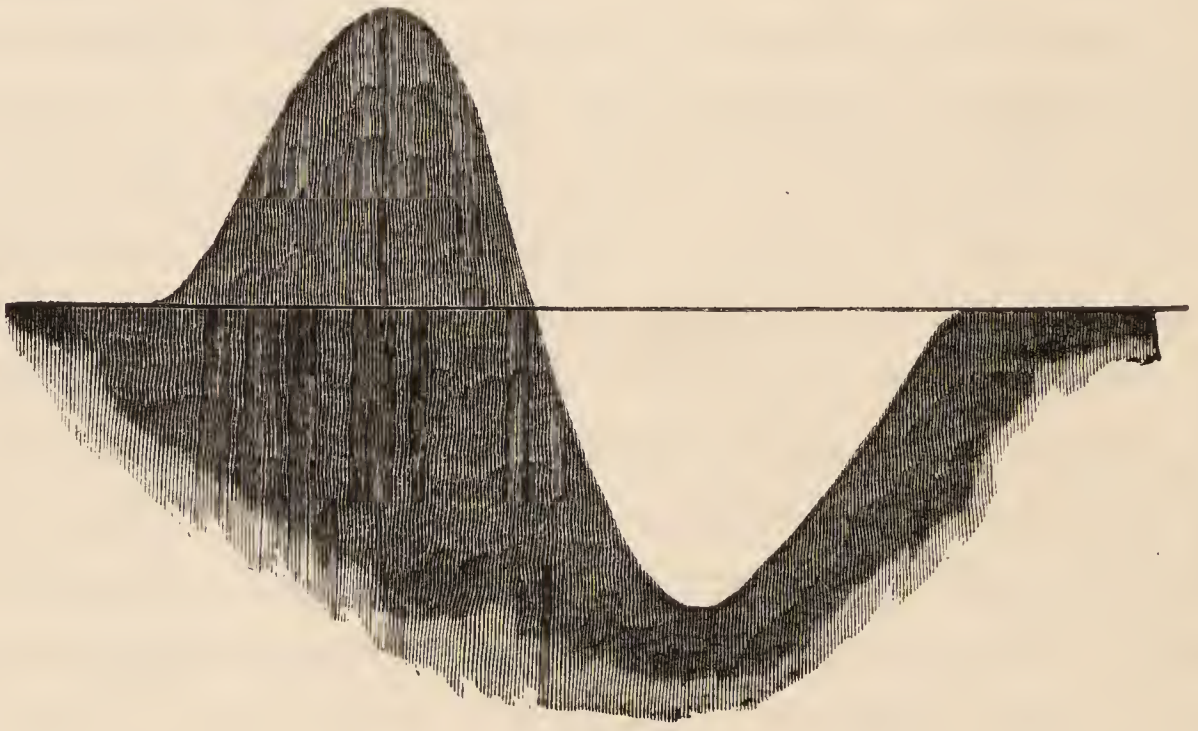

Fig. 3.

If however, the point A is cooled, the negative state there is slowed and diminished in amplitude, so that the two variations would now be represented by Fig. 4.

This would give a triphasic variation (Fig. 5) and this as we saw was one of the stages we obtained when cooling the base of the heart.

But the explanation will not account for the complete reversal

of the capillary curves. For if the wave starts at any point *A* whatever its time-course, it is plain that negativity of *A* will always be the first phase on the photographic curve. Yet we found above that the first wave shewing basal negativity *disappeared entirely* with further cooling of the base. Hence, if we trust the record of our in-

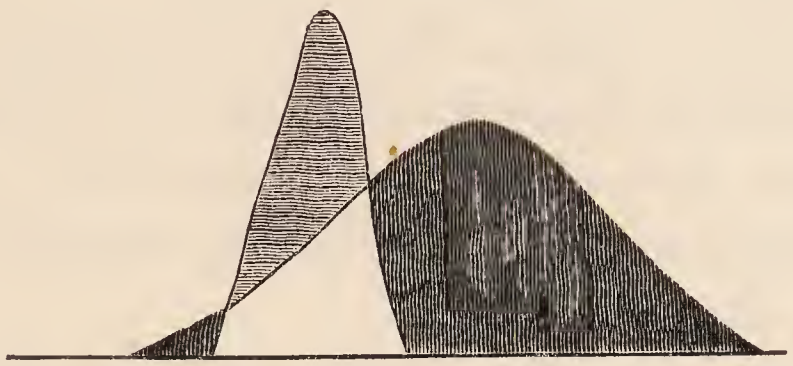

Fig. 4.

strument, we are driven to the conclusion that in the mammalian ventricle, the *excitatory wave is distinct from the wave of negativity*, and may precede it, and that the excitatory wave travels from the auricle to the base, and thence to the apex of the ventricle, so causing under normal circumstances an electrical variation beginning at the base and ending at the apex. Cooling the base would both leng-

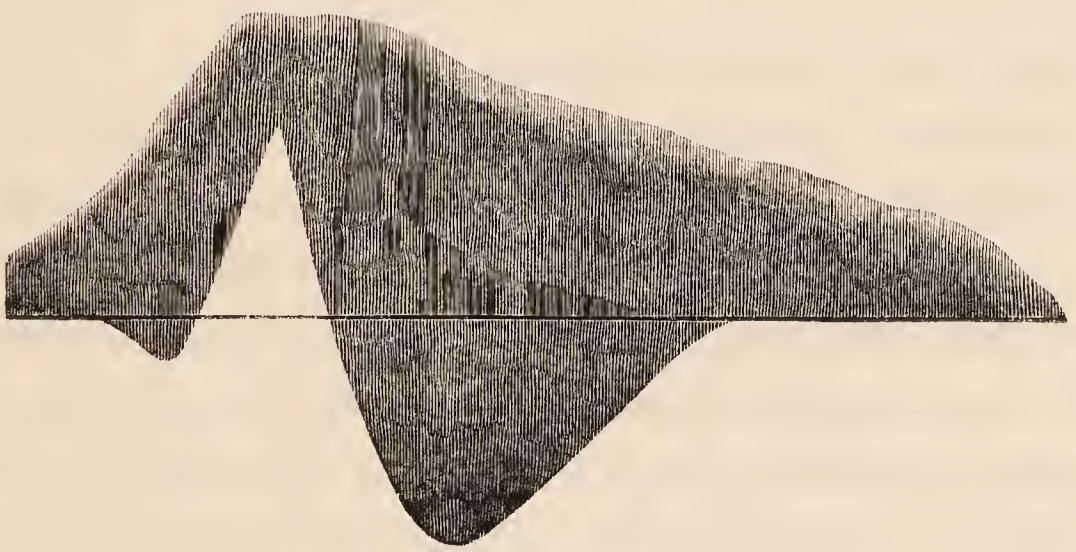

Fig. 5.

then the latent period of the variation and also alter the character of its curve, so that on the electrometer the first effect of cooling the base would be to produce a triphasic variation, and the final effect to reverse the diphasic curve altogether.

We may conceive three ways in which this wave of excitation might travel from base to apex:

a) It might travel in the muscular fibres. This cannot be accepted without much stronger evidence. At present the bulk of evidence seems to show that the wave of excitation in muscle is simultaneous, if not identical, with the wave of negativity.

b) It might be conducted by a deeper layer of muscular tissue. We think the following arguments tell against this view:

1. The effect of using cooled air in respiration must tend to lower considerably the temperature of the blood pouring into the left side of the heart. This will cool the internal layer of muscle so that it will be in a worse condition for conducting excitation than the outer layer which we are leading off.

2. We made many attempts to lead off two points on the internal surface of the left ventricle by means of non-polarizable electrodes at the time when we were still using cold air in respiration, and hence obtained apex negativity preceding basal negativity. The difficulties, however, were so great that we only obtained one photograph presenting a good diphasic variation, and this shews apex negativity preceding basal negativity, (see Plate XV. Fig. 8), so disproving the suggestion of a wave travelling internally from base to apex and then externally from apex to base. The method used in leading off the interior of the heart was the following. (Fig. 6.)

The electrodes consisted of two narrow glass tubes, about three inches long, curved for about half an inch at one end *a* and *b*. The curvature of the basal electrode was more acute than that of the other. These tubes were filled with saturated zinc sulphate solution; the curved ends were plugged with zinc sulphate clay, and just before use a small plug of saline clay was pushed into the ends. An amalgamated zinc wire was dipped into the solution and leakage was prevented by a short bit of india-rubber tubing, connecting the zinc wire and the glass tube; the copper wires soldered to the zinc were well insulated by telegraph cement. Each of these tubes fitted into a block of ebonite, one of which, viz: that for the apex c), had a spring attached to it, armed with a button at its extremity and so arranged as to be exactly opposite the end of the glass tube; the object of the spring being to keep the electrode in contact with the ventricular wall. To

the other piece of ebonite d) was fitted a brass tube carrying a tambour at its extremity; this tambour was provided with a button arranged to be opposite the end of the basal electrode. It will be seen that the latter electrode is an adaptation of Frédéricq's „pince cardiographique." By means of the tambour the beats of the heart were registered at the same time as the electrical variations. The glass tubes were introduced through small incisions in the left auricular appendage, and were tied in, scarcely any blood being lost in the pro-

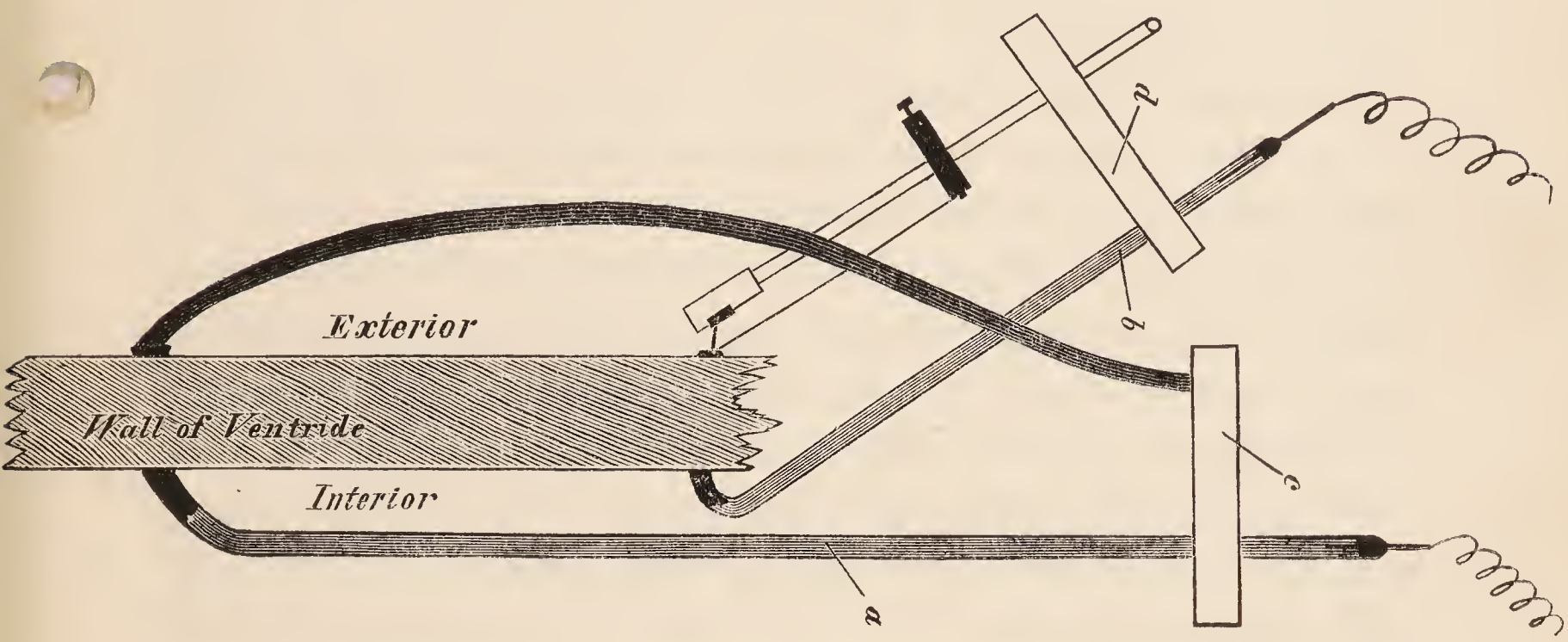

Fig. 6.

cess. They were then carefully pushed into the ventricular cavity and when in position the ebonite blocks were put on to them, the spring and the button of the tambour adjusted to press slightly on the ventricle and keep the electrode in contact with it.

3. The only other path possible for the excitation wave is through nerves, and we think that our experiments suggest the possibility that, under normal conditions, the wave of excitation excited by an auricular contraction travels along a nerve network, from base to apex of the ventricle, and that the muscular fibres are excited from these nerves. Such a nerve network has been shown to exist in ventricular muscle by Openchowski [1]) and others.

---

[1]) Archiv f. Mikros. Anat. Band XXII.

We must nevertheless bear in mind that it is quite possible that there is really an electrical change at the base preceding that at the apex under all circumstances, but that it develops in some cases so slowly that no appreciable movement of the electrometer is caused by it before the development of the large E. M. F. of the warmer apex comes to swamp it; that is to say, that the state of excitation of the muscular elements at the base, is sufficient to excite adjoining elements and thus be propagated along the ventricle before it has attained sufficient intensity to cause the slightest movement of the mercury meniscus of the electrometer.

To return to the normal curve for a moment. It will be noticed at once how different is the appearance of our curve from that of those obtained by Sanderson and Page from the frog and tortoise; yet on consideration we shall see that this difference lies merely in the duration of the electrical change. In the frog's heart, if we lead off from two points, *a* and *b* at apex and base respectively, each contraction is attended by a double variation, the two phases of which are separated by an equipotential interval. At the commencement of each beat, *b* becomes negative to *a*, and the meniscus makes a rapid movement towards the point (if the base electrode be connected with the acid); in a short time the contraction reaches *a*, so that the negativity at *a* and *b* is equal; this is equivalent to putting on an E. M. F. equal and opposite to the first E. M. F., and the mercury returns rapidly to the position of zero potential-difference, so causing what Dr. Sanderson calls a „spike". So long as the ventricle remains contracted as a whole, the meniscus remains in this position; but when relaxation begins at the base there is again a difference of potential between *a* and *b* — *a* now becoming negative to *b* for a short time, and producing a movement of the meniscus stays from the point; and there being no opposing E. M. F. to bring it back, the return of the meniscus to zero is slow; for, the electrometer having to discharge itself through the high resistance of the electrodes and tissues, it follows its characteristic logarithmic curve [1]).

---

[1]) Burch, Proc. Roy. Soc. Vol. 48. p. 91.

In the mammalian heart the duration of the contraction and that of the electrical change is much shorter than in the frog's heart, and the second phase follows the first immediately, no equipotential phase being apparent. It may be made to appear by cooling the heart as a whole, by which means the rate of propagation is slowed and the duration of the electrical change increased. It is possible that if the movement of the meniscus in our electrometer had been sufficiently rapid, we should have found an equipotential interval in all cases. With our electrometer the mercury had not returned to zero before the second phase commenced; in some cases (see Plate XV. figs. 9 and 10) with a small excursion of the meniscus, we have obtained an equipotential interval in the normally beating heart.

There is another circumstance which may have some bearing on this absence of an equipotential interval. The curves given by Sanderson and Page were obtained by artificial excitation of the heart brought to a standstill by a Stannius ligature, whereas photographs of the electrical change of the spontaneously beating heart of frog and tortoise, obtained by one of us (Bayliss) in conjunction with Prof. Schäfer, show no equipotential interval. (Plate XV. figs. 11, 12, and 13.) Whether this was due to the slowness of the electrometer, we cannot say; but it seems to suggest some difference between natural and artificial excitation.

In reference to the effects of heat and cold described above, a few experiments made on the tortoise heart are of interest. Unfortunately we had difficulty in obtaining tortoises, and were unable to repeat these experiments as we should have wished. We cut a strip of ventricular muscle and laid it on the top of a warming and cooling apparatus, similar to that described by Sanderson and Page, and consisting of two brass tubes soldered together, either of which could be warmed or cooled by a current of warm or iced water. The ventricular muscle was led off in such a manner that the neighbourhood of either electrode could be warmed or cooled at will, and it was excited by induction shocks near one electrode. We found that by cooling the electrode nearest the excited point, we could reverse the electrical effect, that is, whilst of course with equal temperature of the two parts,

the curve was diphasic, indicating negativity of the point nearest the excited place preceding negativity of the further electrode, yet when the excited part was cooled, a diphasic curve of the opposite nature was produced, indicating negativity of the points further from the excited point preceding negativity of the point nearer; moreover, it appeared to the eye as if the further half contracted before the nearer (cooled) half. Unfortunately, through lack of material we were unable to repeat this experiment, and would not lay too much stress upon it. (Plate XVI. figs. 14, 15, 16.)

II. Time measurements.

In attempting to make accurate time measurements of the latent period of contraction of the heart muscle, or of the velocity of propagation of the wave of contraction in warm-blooded animals, difficulties are met with which do not occur in working on cold-blooded animals. We cannot take the heart out of the body, or interrupt its blood-supply without its losing at once its normal excitability and soon dying altogether. We cannot use the Stannius ligature to bring it to a standstill, and although we can stop the heart by exciting the vagus, yet the duration of the stoppage must be extremely short, or the blood becomes deoxygenated and affects the heart injuriously; moreover, under vagus excitation the conditions of the ventricular walls as regards tension, are abnormal — the heart becoming enormously distended. Again, excitation of the vagus causes a marked change in the excitability of both auricles and ventricles, in the case of propagation from auricle to ventricle and probably from one part of the muscular wall to another; as a rule, in fact, we found the auricles inexcitable to artificial stimulation under vagus inhibition.

We therefore made use of a method suggested by Prof. Gotch. If any part of the auricle or ventricle be stimulated at a rate slightly above the normal rhythm, the heart will beat regularly in response to each stimulus, and not only will the ventricle follow the auricle, but if the ventricle itself be excited, the auricle will beat in regular

sequence to the ventricular contraction. We were in doubt as to this at first, and therefore took tracings with levers attached to auricle and ventricle, and found it to be unquestionably the case; it is clearly of some importance with respect to the nature of the transmission from auricle to ventricle. (We were unaware at the time that we made these investigations, that this method of producing a reversed beat had been already described by Mc. William [1]).)

By this means then, we replace the natural by an artificial rhythm, and we know also the exact time at which the excitatory process commences. The stimulus used was an induction shock, excited by the break of the primary circuit. This was performed about 3 or 4 times per second by one of the contacts of a v. Frey's rheotome, the other contact being arranged to short-circuit the make-shock.

To measure the latent period of the electrical change of the ventricular muscle, some point of the surface of the ventricle was excited immediately underneath one of the leading-off electrodes.

To measure the velocity of propagation of the wave from auricle to ventricle, the base and apex of the ventricle were led off to the electrometer and the stimulating electrodes applied either to an auricular appendage or to the body of one of the auricles in front or behind.

When using morphinised dogs, it is necessary to cut both vagi, otherwise the vagus excitation produced by the drug acting on the cardio-inhibitory centre, lowers the excitability of both auricle and ventricle, and produces a „block" between auricle and ventricle, so that either the point excited does not respond to every stimulus, or the contraction, if produced, does not travel across the auriculo-ventricular groove.

In these experiments, in addition to the excursions of the mercury meniscus, there were simultaneously photographed by means of two chronographs the moment of stimulation and the vibrations of a tuning-fork of 100 per second.

In order to obtain more exact information of the time-course of

---

[1]) Journal of Physiology. Vol. IX. 1888.

the electrical change, we attempted to use the repeating rheotome both with galvanometer and electrometer; but found it impossible, owing to the fact that the effect of each stimulus is not always precisely equal to that of the one preceding it, so that the movements of the galvanometer-needle and of the meniscus of the electrometer were too irregular to afford any information whatever.

Latent period of ventricular and auricular muscle.

This was found to be so short, in cases of direct excitation, that we could not measure it. The light used (lime-light) was not powerful enough to enable us to give a sufficiently rapid rate of movement to our photographic plates; we could, in fact, obtain no image when our rate of movement exceeded 10 centim. per second. The latent period of the electrical change is certainly not more than .005″.

Velocity of propagation from auricle to ventricle.

If we led off the base and apex of the ventricle and excited some point of the auricle, we found that a considerable period of time (.12″ to .16″) elapses before there is any electrical variation in the ventricle. This delay occurs chiefly in the passage of the wave from auricle to ventricle, for we found that it made very little difference in the time required, whether we excited the auricles at the tip of an appendage or close to the auriculo-ventricular groove, or again, whether we led off from two points near the base or from two points near the apex of the ventricle. Thus, in one experiment, when the tip of the right auricular appendage was excited, the time that elapsed before the electrical change occurred at the base of the ventricle was .14″; the auricle was then excited, close to the auriculo-ventricular groove and the time of propagation was now .13″. The numbers obtained in various experiments were the following.

a) Exciting appendages and leading off base and apex:
0.13″, 0.12″, 0.14″, 0.13″, 0.13″, 0.12″.

b) Exciting auricle near groove:
0.13″, 0.14″, 0.16″.

The last number was obtained when exciting the middle of the posterior surface of the auricles.

As the heart dies, the block between auricle and ventricle rapidly increases, till at last it becomes complete, and no transmission of excitation from auricle to ventricle takes place. In one case, in which we measured this period of time in the excised heart in the warm chamber immediately after removal from the body, we found that the excitatory change took 0.30″ to travel from auricle to base of the ventricles.

Velocity of propagation in ventricle.

Sanderson and Page showed that the velocity of propagation of the electrical change in the frog's ventricle, might be measured by the time-interval between the beginning and culmination of the initial phase of the variation. Our observations on Mammals have shown, however, that in warm-blooded animals the duration, and indeed the very existence of this initial phase, are so extremely susceptible to slight changes of temperature that no measurement of it could be relied on as giving the real time taken in the passage of the excitatory change from base to apex of the ventricle. Probably we should obtain a more accurate estimation of this time-interval in intact animals, where the conditions as to temperature are constant. (see Appendix.)

For examples of the kind of curves obtained in these measurements, see Plate XVI. figs. 17 to 21.

No definite conclusions as to the nature of the transmission from auricle to ventricle can be drawn from these results. It may be said that the loss of time at the groove, and the fact that transmission is possible in either direction across the groove, point to a direct muscular continuity. Anatomists, however, state the contrary, but until a careful series of sections has been made, the point cannot be considered decided. There is, moreover, the possibility of a nerve-network of a rudimentary kind, and we must not be too dogmatic as to the impossibility of such a network taking up excitation at any point and transmitting it to any other point. That nerve-fibres of the ordinary kind are not the means of transmission is, we think, shown by our results.

III.

The supposed tetanic character of the ventricular contraction.

Frédéricq has come to the conclusion that each ventricular contraction is composed of 3 or 4 single twitches fused together and is therefore of the nature of a tetanus. He bases his conclusions on the following evidence.

1. The vibrations on the plateau of the ordinary cardiographic curve, and of the tracings of intraventricular pressure, obtained by Chauveau and Marey.

2. On the descending parts of the electrometer curves (mostly monophasic), obtained by him from the heart in situ, are to be observed small superposed vibrations, indicating the compound nature of the electrical change.

3. If the aorta be ligatured and the *heart* excised and the electrical variation of the dying heart photographed, some of the beats, just before the heart goes into delirium, show two or three summits to the electrical curve.

We have found no evidence that the cardiac contraction is tetanic; on the contrary, we have, we venture to think, established the fact that each contraction of the mammalian ventricle is a single wave, starting normally at the base and spreading thence over the whole substance of the ventricles.

1. With regard to Frédéricq's first argument, we have no right to speak, not having made any special researches on the subject. But we would suggest that the superposed curves on the systolic plateau of an endocardiac pressure tracing might be equally well attributed to the elastic vibrations of the ventricular and aortic walls, induced by the sudden increase of pressure in their cavities, reinforced and exaggerated by the periodicity of the recording instruments employed.

2. The small superposed vibrations on the descending parts of the electrometer-curves taken from the heart beating *in situ* are clearly due to vibrations of the apparatus itself, and not to any cardiac event, for if we look closely at his figures, we see that they are as

clearly marked on the flat part of the photograph between the heart beats as on the curve of the variation caused by each heart beat — unless we are to take these vibrations as shewing the existence of a tetanic relaxation of the mammalian heart.

3. Frédéricq however lays most stress on the three-topped curve obtained from dying hearts, or rather from hearts just before they go into a condition of delirium. The fact that these curves are monophasic shews that one of the leading-off points must have been injured. In the next section we shall consider certain forms of curves which we call „stepped" curves, and which are obtained when one of the leading-off points is injured. The examples we give are very similar to the two-topped curves given by Frédéricq. We have also obtained three-topped curves in some cases, in which the heart, which had been beating regularly and giving a two-topped or stepped curve, suddenly gave two or three double beats. Just before the heart goes into delirium, it beats extremely irregularly, the beats often consisting of pairs of partially fused contractions; and we feel inclined to ascribe a like origin to the three-topped curves of Frédéricq.

But in whatever way we explain these curves, we do not feel that one is justified in drawing conclusions as to the nature of the normal beat from the electrometer-curve of the electrical variation of a dying and injured heart on the point of going into delirium cordis.

IV.

It remains to mention a few facts relating to the *effects of injury* on the form of the electrical wave. As shewn by Sanderson and Page, the effect of injury of the muscular substance at either of the points led off, is to diminish or abolish the excitatory state at that point, hence leading to the production of a monophasic electrical variation when the excitatory state is quite abolished at one contact, or to various forms of what we may call „stepped" curves when it is diminished. The monophasic electrical variation is the expression of the

negativity of the uninjured points and lasts during the whole period of contraction of that point. As an example of a „stepped" curve we give fig. 22, Plate XVI which can be explained as below, Figs. 7 and 8.

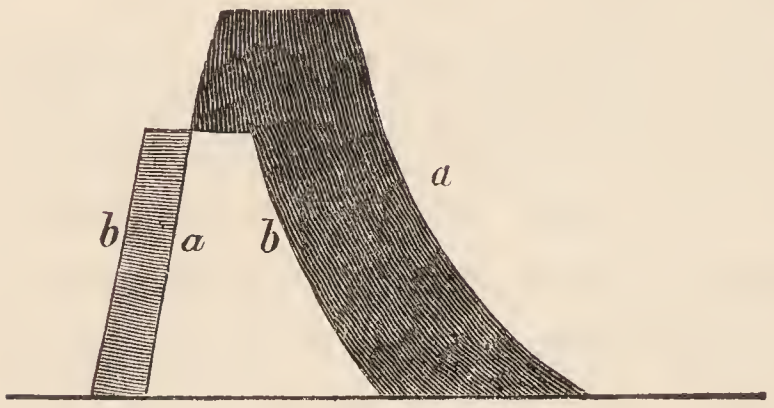

Fig. 7.

It is perhaps necessary to meet a possible objection to our interpretation of some of the curves. It might be said that in the triphasic curves we have mistaken negativity of the auricle for negativity of the base of the ventricle. To meet this objection, we have taken photographs of the heart variation, leading off at one time apex and auricle

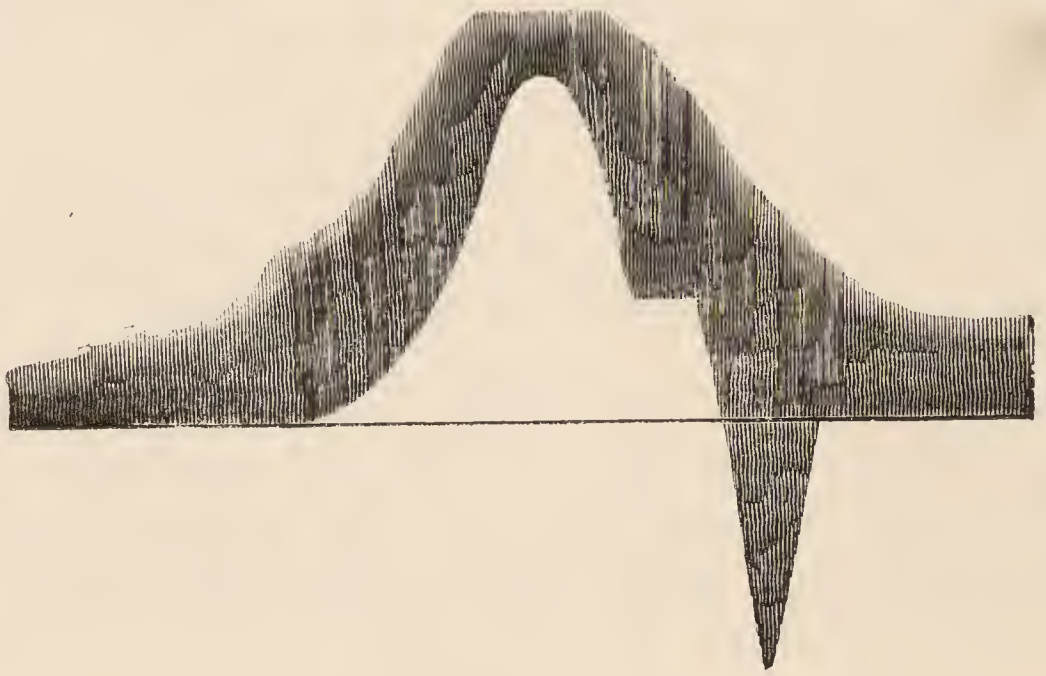

Fig. 8.

and at another time base and apex, and could detect no difference between them; in fact, with our electrometer the electrical change of the auricle was too small to produce any movement of the meniscus.

General summary of results.

1. Our experiments show that in general, the conclusions of Sanderson and Page with respect to the heart of the cold-blooded vertebrate, can be applied also to that of the mammal.

2. The ventricular contraction is a single wave, starting from

the base, at the auriculo-ventricular groove, and spreading thence to the apex.

3. There is a considerable block at the auriculo-ventricular groove to the transmission of excitation from auricle to ventricle.

4. The effect of using cold air in respiration, or cooling the base of the ventricles by other means, is to reverse the electrical changes, causing a wave of negativity which apparently starts from the apex.

5. The effect of injury is similar to that observed by Sanderson and Page in the frog, viz., abolition or diminution of the state of excitation at the injured part.

Appendix.

On the Electromotive Changes connected with the cardiac Beat in Man and the Dog with chest unopened.

(From experiments made in the Physiological Laboratory of the University of Oxford.)

To Waller is due the credit of having first discovered the possibility of recording and studying the electromotive changes attending the cardiac contraction in man and the intact animal. In his first paper on the electrical change of the heart of man [1]), we find that he observed on the electrometer two movements of the meniscus in the same direction accompanying each beat, and that they were in such a direction as to indicate negativity of the base to the apex occurring twice in each beat; but no explanation is given. In a subsequent paper [2]) Waller gives a different account of the variation, having taken photographs of the variation which seemed to show that negativity of the apex *preceded* that of the base.

In the paper to which this is an appendix, it is shewn that in the dog's heart, in as normal a condition as possible when exposed, negativity of the base always precedes that of the apex, and the occurrence of apical negativity preceding basal negativity is explained as due to local cooling of the base.

---

[1]) Journal of Physiology. Vol. VIII. p. 229.

[2]) Phil. Trans. Vol. 180. 1889. p. 169—194.

It was clearly of importance, therefore, to repeat the observations of Waller on the intact animal, and as the capillary electrometer at our disposal at University College, London was not sufficiently delicate to show so small a variation, by the kindness of Prof. Burdon-Sanderson we were enabled to make use of the apparatus in his laboratory at Oxford. We had also the advantage of Prof. Gotch's assistance in carrying out the observations, and beg him to accept our grateful acknowledgments.

It is unnecessary to describe in detail the apparatus used, it being essentially similar to that described above. An important advantage, however, it had, in that we were able to use the electric arc light in place of the lime-light, and hence greater magnifying power on the projecting microscope. The method of leading off, found to give the best results, was to place our electrode, consisting of a thick rod of amalgamated zinc having a sponge soaked in brine tied over the end, on the situation of the apex beat; the other electrode consisting of a similar zinc rod dipping into a basin of brine in which the right hand was immersed.

When observed by the eye, the movement of the meniscus appeared to consist of two phases, the first short and sharp, and the second more prolonged; the direction of the second was easily made out, but that of the first it was impossible to decide by mere inspection; in fact, different observers had different opinions. A photograph, therefore, was the only possible way to decide; indeed, when leading off apex beat and epigastrium on one occasion, we thought we obtained a movement showing that the apex became negative first; but a photograph showed that we had completely missed the first phase of base negativity.

We led off from various parts of the body, but found that apex beat and right hand gave the largest excursion, the photograph of which in the case of one of us (E. H. S.), is reproduced in Plate XVII, fig. 23 (to be read from right to left); the uppermost of the three curves is the chronograph tracing, marking $^1/_{10}$ secs., the middle one is the tracing of the carotid pulse, obtained by means of a tympanum, and the lowest one is the movement of the electrometer me-

niscus. This latter is seen clearly to consist of three phases: 1st., a „spike" towards the point of the capillary; 2nd., a more prolonged excursion away from the point; and, 3rd., a large and prolonged movement towards the point again. Now the connections of the electrometer were such that a movement towards the point indicated negativity of the electrode nearest the base of the heart: the curve therefore shows that the *base becomes negative before the apex*, and that its negativity outlasts that of the apex; apical negativity being indicated by movement away from the point. Our photographs then confirm the first account given by Waller of the variation in man, there being two excursions of the meniscus in the same direction accompanying each beat.

We have led off and photographed the variations from the various points mentioned by Waller; but found invariably that the electrode nearest the base became negative before that nearest the apex.

Fig. 24 of Plate XVII gives the variations from apex beat and right hand in another person (W. M. B.), and is given to show that here also, movement towards the point (i. e., base negativity) precedes apex negativity; the latter in this case being shewn by the return of the sharp spike to the base line before the commencement of the third phase.

Fig. 25 gives the variation in the dog under morphia, led off from apex beat and right forepaw. It also is of the same triphasic nature, the base becoming negative before the apex, and remaining negative longer.

That the normal variation in the intact animal should be triphasic was rather surprising to us, as we had learned to look upon this form of curve in the exposed heart of the dog, as due to local cooling of the base. The question suggests itself whether this form of the electrical variation may be due to the proximity of the apex to the warm liver. Against this view is the fact that we found that placing the apex electrode in the dog between the liver and diaphragm, and hence separating the heart from contact with the liver, did not affect the triphasic nature of the variation. Probably therefore, we

must look upon it as indicating that the excitatory state at the base lasts longer than at the apex.

The total duration of the electrical change in man as measured on the photograph of the plate, is about 0.35″; in the dog, rather less, about 0.30″. The numbers given by Landois for man [1]) are

Mean duration of ventricular contraction	.192″
Mean duration of maintenance of contraction	.082″
Mean duration from beginning of relaxation to closure of semilunar valves	.072″
Mean duration from closure of semilunar valves to beginning of pause	.200″
Total	0.546″

If we take the first three together as indicating systole of ventricle, as Donders does, the number is 0.346″, which agrees very closely with our numbers.

It will be observed on measurement, that the distance of the dicrotic wave in the carotid pulse of fig. 23 from the beginning of the beat is equal to that of the culmination of the third phase from the beginning of the electrical change, that is, according to our interpretation it occurs at the time when the excitatory state at the apex ceases.

The *rate of transmission*, as Sanderson and Page have shewn, can be measured by the time from the commencement to the culmination of the initial phase; this is too short in our curves to be measured with accuracy, but it appears to be about 0.02″, which gives a velocity of 5 metres per second, assuming the length of the ventricle to be traversed as 10 centimetres.

It is worthy of note that the direction of the electrical change of the heart beating slowly and normally, is considerably greater than that of the dog's heart, beating 3 times per second under artificial excitation from auricle. If we compare fig. 23 Plate XVII (the heart of man) with fig. 19 Plate XVI we notice the similarity of the

[1]) Centralblatt f. d. medic. Wissensch. 1866. p. 179.

electrical variation; but in the latter case the total duration is only 0.18″, in the former 0.35″, and in the intact dog 0.30″.

We conclude then, that the electrical variation in man and in the intact dog, begins with negativity of the base. This is followed by a phase in which negativity at the apex predominates, and this again by a third phase, consisting of negativity of the base; therefore in man as in the exposed heart of the dog, the ventricular contraction begins at the base and travels to the apex in the form of a wave, but in the former case the excitatory state at the base outlasts that at the apex.

Description of the Plates XV—XVII.

Plate XV.

All curves to be read from left to right.

Fig. 1, 2, 3. Different forms of the diphasic normal curve. Apex and base led off. Warmed air in respiration. Base negativity precedes apex negativity. Time-trace $^1/_8$″. (Base to acid. Apex to capillary.)

Fig. 4. Normal curve. Warmed air. Base negativity followed by apex negativity. Time $^1/_8$″. (Base to acid. Apex to capillary.)

Fig. 5. Same heart, warmed air replaced by cold, transition stage.

Fig. 6. Same heart, further action of cold in causing complete reversal, apex negativity followed by base negativity.

Fig. 7. Same heart, warm air replaced. Return to original condition of base negativity followed by apex negativity.

Fig. 8. Base and apex of inner surface of left ventricle led off. Cold air. Apex negativity followed by base negativity. Time-trace $^1/_8$″. (Base to acid. Apex to capillary.)

Fig. 9. Curve showing short equipotential interval. (Base to capillary. Apex to acid.)

Fig. 10. Curve with longer equipotential interval. Time-trace in both. $^1/_8$″. (Base to acid. Apex to capillary.

Fig. 11. Variation of spontaneous beats in frog heart, to show absence of equipotential interval. (Connections as in 10.)

Fig. 12 and 13. Similar curve from tortoise ventricle. Time-trace in seconds. (Connections as 10.)

Plate XVI.

All curves to be read from left to right.

Fig. 14. Strip of tortoise ventricle cooled at both ends, showing negativity of stimulated end preceding that ot more distant end. Time-trace $^1/_8$″. (Stimulated end to capillary.)

Fig. 15. A similar preparation warmed at end distant from stimulation, cooled at stimulated end, showing a triphasic variation produced as described under figs. 4 and 5 in text. Time-trace $^1/_8$″.

Fig. 16. A similar preparation, showing complete reversal of variation produced by cooling stimulated end and warming more distant end, showing negativity of more distant end preceding that of directly stimulated end. Time-trace $^1/_8$″.

Fig. 17. Left auricle and apex behind led off. Auricle excited under electrode. Latent period 0.12″. Time-trace $^1/_{100}$″.

Fig. 18. Base and apex led off. Right auricular appendage excited. Latent period 0.12″. Time-trace $^1/_{100}$″. (Base to acid.)

Fig. 19. Base and apex led off, base to acid, apex to mercury in capillary. Left auricular appendage excited. Latent period 0.12″. Time-trace $^1/_{100}$″.

Fig. 20. Base and apex led off, excited $^1/_4$ in. from base electrode. Latent period not more then 0.01″. Time-trace $^1/_{100}$″.

Fig. 21. Excised heart in warm chamber. Apex and base of right ventricle *behind* led off. Right auricle excited, it responded to excitation. Latent period 0.20″. Time-trace $^1/_{100}$″. (Base to acid.)

Fig. 22. „Stepped" curve. Excised heart in warm chamber, beating spontaneously. Time-trace $^1/_{100}$″. (Base to acid.)

Plate XVII.

All curves to be read from right to left.

Fig. 23. Electrical variation of human heart (E. H. S.) led off from apex beat and right hand. Middle curve is carotid beat. Time-tracing $^1/_{10}$″. Apex beat to capillary. Rt. hand to acid.

Fig. 24. Similar curve from another heart. (W. M. B.) Apex to acid. Hand to capillary.

Fig. 25. Similar curve from dog under morphia. Led off from apex beat and right fore-paw. Connections as in Fig. 24.

Printed by Richard Hahn, Leipzig.